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# Predictors of human PBDE body burdens for a UK cohort

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## 1Abstract

2 Human exposure to polybrominated diphenyl ethers (PBDEs) was investigated in a cohort  
3 of 20 UK adults along with their anthropometric covariates and relevant properties such as room  
4 surveys, lifestyle, diet and activity details. Selected PBDE congeners were measured in matched  
5 samples of indoor dust, (n=41), vehicles (n=8), duplicate diet (n=24), serum (n=24) and breast  
6 milk (n=6).

7 Combined exposure estimates via dust and diet revealed total PBDE intakes of 104 to  
8 1,440 pg kg<sup>-1</sup> bw d<sup>-1</sup> for ΣBDEs<sub>3-7</sub> and 1,170 to 17,000 pg kg<sup>-1</sup> bw d<sup>-1</sup> for BDE-209. These adult  
9 intakes are well within health reference doses suggested by the European Food Safety Authority  
10 (EFSA) and the US EPA. For this cohort diet was the primary source of intake of BDE<sub>3-7</sub> congeners  
11 for the majority of the cohort, with dust the primary source of BDE-209. Primary sources of PBDE  
12 exposure vary between countries and regions with differing fire prevention regulations.

13 Estimated infant exposures (ages 1.5 to 4.5 years) showed that BDE-99 intake for one of the  
14 households did not meet EFSA's recommended margin of exposure, a further two households  
15 were borderline for high level dust and diet intake.

16 Males and those having a lower body fat mass had higher serum BDE-153. Higher meat consumption  
17 was significantly correlated with higher BDEs<sub>3-7</sub> in serum. A reduction in dietary BDEs<sub>3-7</sub> would  
18 therefore result in the greatest reduction in BDE-99 exposure. Rooms containing PUF sofas or  
19 armchairs over 20 years old had higher BDEs<sub>3-7</sub> in their dust, and rooms with carpets or rugs of that  
20 age had higher dust BDE-209. Dusting rooms more frequently resulted in significantly lower  
21 concentrations of all major congeners in their dust. Correlation between BDE-209 body burden and  
22 dust or diet exposure was limited by its low bioaccessibility. Although vehicle dust contained the  
23 highest concentrations of BDEs<sub>3-7</sub> and BDE-209, serum BDEs<sub>3-7</sub> correlated most strongly with  
24 bedroom dust.

25

## 261 Introduction

27UK residents are still exposed to a class of potentially harmful brominated flame retardants,  
28polybrominated diphenyl ethers (PBDEs), even though European Union regulations  
29restricting their manufacture, use and importation came into force in 2004 and 2008. Since  
30the 1970s PBDEs have been incorporated into fabrics, foam cushioning and plastics used in  
31everyday items such as vehicles, soft furnishings and electronics. PBDEs slow the rate of  
32ignition and fire growth in petroleum based polymers and resins. PBDEs are not chemically  
33bonded to these materials and are emitted into indoor dust and air through use and  
34volatilisation (Rauert and Harrad, 2015; Sjödin et al., 2003). They can then move into the  
35wider environment where they have been found in sewage sludge, soils and river and lake  
36sediments (Allchin et al., 1999; De Boer et al., 2003; Eljarrat et al., 2008; Harrad et al., 2009).  
37They are persistent organic pollutants as defined by the United Nations Environment  
38Programme's Stockholm Convention and have an environmental half-life of several years.  
39They can travel long distances in the atmosphere and are lipophilic, concentrating in animal  
40and marine fats. These qualities and their wide usage have led them to permeate  
41environments and food chains around the world (Fromme et al., 2016).

42A recent systematic review of human health consequences of exposure to PBDEs concluded  
43health effects may include thyroid disorders, reproductive health effects, and  
44neurobehavioral and developmental disorders (Kim et al., 2014). Evidence of these effects  
45has been seen in animal and *in vitro* research, where the mechanism appears to be altered  
46hormone regulation (endocrine disruption) (Linares et al., 2015; Marchesini et al., 2008;  
47Meerts et al., 2000; Viberg et al., 2006). Exposure during key developmental stages in  
48infancy is most damaging as this is the time when altered hormone regulation will have the  
49greatest impact. Recent estimates of the economic cost of just the intelligence quotient (IQ)  
50points loss and intellectual disability due to PBDE exposure was \$266 billion in the USA and  
51\$12.6 billion in the EU (Attina et al., 2016). These figures must be balanced against amounts  
52saved due to fire prevention resulting from furnishing flammability standards e.g. £140  
53million annual savings in the UK estimated by prevention of death, injury and damage to  
54property as a result of Furniture and Furnishings Fire Safety Regulations 1988 that require

55use of flame retardant chemicals. (BIS, 2009). PBDEs were only one group of flame retardant  
56chemicals from the several BFR groups commonly used to meet such regulations.

57In 2004, use of two commercial PBDE products, Penta-BDE and Octa-BDE, were restricted  
58within the EU (European Council Directive 2003/11/EC) and voluntarily phased out in the  
59USA. In 2009, they were added to the Stockholm Convention list of POPs for elimination.  
60Penta-BDE had been primarily used in polyurethane foam (PUF) in soft furnishings, vehicles  
61and printed circuit boards, in greatest amounts in the USA. Furnishings could contain one to  
62four percent Penta-BDE to comply with fire safety regulations (Hammel et al., 2017). The  
63Octa-BDE commercial product has been produced and used less widely than Penta-BDE. Its  
64major use has been in acrylonitrile-butadiene-styrene (ABS) plastics, such as electronics and  
65resin casings of office equipment. The Deca-BDE commercial product has been added to  
66furnishing textiles, and in high impact polystyrene (HIPS) for cables, sockets, mobile phones,  
67fridges and TV housings.

68Concentrations of BDE-209 are higher in UK indoor dusts than in dusts from mainland  
69Europe (Frederiksen et al., 2009; Harrad et al., 2008b) as a result of the UK's more stringent  
70fire safety regulations ([Furniture and Furnishings Fire Safety Regulations 1988/1989, 1993](#)  
71[and 2010](#)). Deca-BDE has been restricted from use in electrical and electronic equipment in  
72the EU since 2008 and was added to Annex A of the Stockholm Convention list of POPs in  
732017. Both diet and contact with indoor dust constitute important exposure pathways for  
74PBDEs (Abdallah and Harrad, 2014). Foods from higher up the food chain, of animal origin,  
75with a higher fat content (i.e. fish), meat and dairy have higher PBDE concentrations (EFSA,  
762011). PBDEs will be circulating in our food chains for many years to come (Harrad and  
77Diamond, 2006), and will be re-circulated back into homes as a result of plastics recycling  
78(Samsonek and Puype, 2013) .

79Whether dust or diet is the primary exposure source for an individual depends on a number  
80of factors; loading of PBDE in dust or food items and the amounts ingested, whether and  
81when PBDE technical products have been phased out in that country and on the age of the  
82individual (Bramwell et al., 2016a). PBDE intake via ingestion and inhalation of dust is the  
83major exposure route for young children in the USA that have frequent hand to mouth  
84behaviours and spend lots of time on floors and carpets (Stapleton et al., 2012). Foetal  
85exposure in the womb and transfer of PBDEs from mother to child during breastfeeding are

86key exposures for children during important developmental periods. For countries outside  
87of the US and Canada, the largest contribution to tri-hepta BDE body burden is thought to  
88be from diet, especially in regions where Penta-BDE use has been restricted for longer. Dust  
89is likely to be most important contributor to exposure to higher brominated congeners in all  
90regions (Sahlström et al., 2015).

91The aim of this study was to determine the major dust and diet sources of PBDEs for a north  
92east England cohort and to consider any potential health risks. The five specific objectives  
93were: (a) to measure PBDE concentrations in dust from homes, work places and vehicles, (b)  
94to calculate relative intake of PBDE via dust in the microenvironments, (c) to evaluate the  
95relative importance of PBDE exposure via indoor dust versus dietary PBDE exposure, (d) to  
96compare intake estimates with reference health values, (e) to investigate relationships  
97between matched environmental and biomonitoring data, and (f) to determine the most  
98effective means of reducing PBDE exposure for the cohort.

99

## 1002 Materials and Methods

101We used a cross sectional and purposive sampling strategy to provide a snap shot of PBDE  
102exposures and body burdens for individuals with expected high, average and low exposures.  
103By comparing individuals with expected divergent exposures, we aimed to reveal the factors  
104influencing body burdens.

### 1052.1 Volunteer recruitment

106We targeted individuals with a range of occupations and diets; such as workers in  
107electronics, soft furnishings, transport, office workers, outdoor workers, oily fish eaters,  
108omnivores and vegetarians. In 2010/11, following ethical approval for the study, volunteers  
109over 18 years of age and with six months or more of domestic and occupational stability  
110were recruited via local authorities, universities, businesses, hospitals, playgroups and  
111breast-feeding groups. A short pre-screening questionnaire was used to identify volunteers  
112that could provide the optimum range of exposures. 79 couples completed the pre-  
113screening questionnaires, 10 couples were invited, and agreed, to participate in the full

114study week. Further description of the cohort is provided in the [Supplementary Information](#).  
115Volunteers gave written informed consent prior to participation.

## 1162.2 Timing of sample collection

117Participants undertook a 'sampling week' during which they completed an exposure and  
118food frequency questionnaire (FFQ), food- and activity-diaries, room surveys including  
119contents, usage and cleaning information and they were asked not to vacuum or dust their  
120home. We adapted the validated WHO-IARC EPIC semi-quantitative dietary questionnaire  
121for the study. On the seventh day of their sampling week, participants collected their  
122duplicate diet samples (DD), and the researcher visited that evening to collect the DD  
123samples, home and vehicle dust samples, questionnaires and surveys. The participants then  
124fasted until their blood sample collection appointment the following morning where  
125anthropometric measurements were also taken. Two couples repeated the full sampling  
126week, with sampling points 6.5 and 7.5 months apart. This provided a longitudinal  
127dimension to the study and an element of validation. All sampling weeks took place  
128between April 1<sup>st</sup> 2011 and 28<sup>th</sup> February 2012.

129

## 1302.3 Serum, breast milk and duplicate diets

131Study participants collected an equal amount of whatever food they ate throughout the day  
132in a contaminant free (verified by tests carried out prior to sampling) lidded polypropylene  
133container for the 24 hour duplicate diet collection. The next day they provided a fasted 60  
134ml blood sample at the Clinical Research Facility of the Royal Victoria Infirmary in Newcastle.  
13550 ml breastmilk samples were collected by either pump or manual expression up to 12 h  
136before and 24 h after provision of the blood sample and kept in pre-cleaned Nalgene  
137containers. Samples were stored at -18°C until transfer to the laboratory for analysis. Details  
138of the serum, human milk and duplicate diet sample collection and analysis have been  
139published previously (Bramwell et al., 2014; Bramwell et al., 2017).

## 1402.4 Dust samples

141Participants were requested not to vacuum or dust their home or vehicle during the  
142sampling week. Dust samples from main living areas (n=11), bedrooms (n=12), and vehicles



143(n=8) were collected by a researcher following a standard sampling protocol to allow direct  
144comparison with previous studies (Abdallah and Harrad, 2009; Coakley et al., 2013; Harrad  
145et al., 2008a; Harrad et al., 2008b). Samples from workplaces (n=10) were collected during  
146the sampling week at the participants' (and their employers') convenience. Dust samples  
147were extracted and analysed at the University of Birmingham, UK, using previously  
148published methods for preparation, extraction, clean up, analysis and quality control  
149(Abdallah et al., 2009; Harrad et al., 2008a; Harrad et al., 2008b). Further details of the dust  
150sample collection, preparation, extraction and analysis are provided in the [Supplementary](#)  
151[Information](#).

## 1522.5 QA/QC

153For the analysis of serum, breast milk and duplicate diet samples, the performance  
154characteristics of the methodology, including quality assurance parameters such as limits of  
155detection (LODs), precision, linear range of measurement, recoveries etc. are included in the  
156previous reports (Fernandes et al., 2008; Fernandes, 2004). Further confidence in the data  
157is provided by regular and successful participation in laboratory proficiency testing and  
158inter-comparison schemes such as POPs in Food 2011 and 2012. PBDEs with IUPAC  
159numbers 17, 28, 47, 49, 66, 71, 77, 85, 99, 100, 119, 126, 138, 153, 154, 183 and 209 were  
160measured. The congeners selected for analysis are those for which reference standards are  
161available. Typical LODs were 1 to 20 ng kg<sup>-1</sup> lipid for PBDEs.

162For the dust sample analysis the average blank (including field blanks) plus 3 standard  
163deviations was used for the limit of detection giving an average 0.7 ng g<sup>-1</sup> for BDEs<sub>3-7</sub> (range  
1640.2-1.7) and 52 ng g<sup>-1</sup> for BDE-209. The PBDE <sup>13</sup>C labelled internal standard recoveries were:  
165<sup>13</sup>C-BDE 47 = 69 ± 20%, <sup>13</sup>C-BDE 99 = 70 ± 20%, <sup>13</sup>C-BDE 153 = 69 ± 20% and <sup>13</sup>C-BDE 209 = 17  
166± 6%. The low recovery for BDE-209 indicates uncertainties in its measurement which are  
167presented here with that caveat. Measurement of SRM NIST 2585 had range 78% (BDE-47)  
168to 122% (BDE-49) and mean 100% of the certified contents.

## 1692.6 Exposure Assessment

170Concentrations of the PBDEs detected in milk and serum samples were lipid-adjusted to  
171allow comparison with the literature. PBDE intake for the 24 hrs of the duplicate diet  
172collection was measured using whole weight duplicate diet PBDE concentrations multiplied

173by the mass of DD collected and divided by the weight of the participant to give  $\text{pg kg}^{-1}$  body  
174weight  $\text{day}^{-1}$ .

175PBDE intakes via dust were estimated by combining measured dust PBDE concentrations  
176with occupation time for individual's various microenvironments (taken from their activity  
177diary) using both average (20 mg/ day) and high (50 mg/day) adult dust intake rates average  
178and high adult dust ingestion as estimated by Jones-Otazo et al. (2005). Although dust  
179ingestion rates may differ between microenvironments and activities (as well as individuals),  
180for the purpose of this study, we have assumed that that dust ingestion occurred pro-rata to  
181the proportion of time spent in each microenvironment during the study week. This was  
182considered the only practical approach in the absence of data to confirm any differences  
183(Abdallah and Harrad, 2009). For time periods when participants were in their home but  
184not in one of the microenvironments measured, the median of their home dust PBDE  
185concentration was used. For time periods when they were in an indoor environment but not  
186in their own home the median of all dusts collected for the study was used. Time spent  
187outside was not assigned a PBDE concentration. Intake rates via dust were divided by the  
188participant's weight to give  $\text{pg PBDE intake kg}^{-1}$  body weight  $\text{day}^{-1}$ .

189PBDE intakes for average and high dust intake scenarios: average  $20 \text{ mg d}^{-1}$ , high  $50 \text{ mg d}^{-1}$   
190(Jones-Otazo et al., 2005) and diet intakes determined from the 24 h duplicate diet  
191concentrations were added together for comparison with the European Food Safety  
192Authority's (EFSA) chronic human daily dietary intake estimations to determine the margins  
193of exposure (MOEs). As PBDE exposure during infancy is considered to present a greater risk  
194to health than that for adults, estimated average and high exposure scenarios for infants  
195aged 1.5 to 4.5 years old were developed as well. Daily average ( $50 \text{ mg d}^{-1}$ ) and high ( $200 \text{ mg}$   
196 $\text{d}^{-1}$ ) dust intake estimations (Jones-Otazo et al., 2005) per kg body weight were extrapolated  
197from individual adult intake values determined for the study. These were added to average  
198and high dietary PBDE intake estimations from the UK total diet study (TDS) (2012) data for  
199infants aged 1.5 to 4.5 years old. Risk assessment for infants from PBDE in breast milks  
200collected for the study has been previously reported (Bramwell et al., 2014).

## 2012.7 Data Analysis

Associations between PBDE concentrations and intakes and potential predictors were explored with scatter plots, box plots and correlations using IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp, Minitab 17 and Excel (Microsoft Office 2013). The distribution of PBDEs in the different matrices was assessed using Shapiro–Wilk statistic. As the majority of distributions were not normal, non-parametric Spearman’s ranking correlation coefficients were determined. The criteria of  $\alpha = 0.05$  for statistical significance was used. A one sample t test was used to compare PBDE intake of omnivorous participants as determined by duplicate diet collection and similar data collected by Harrad et al. (2004) to investigate any temporal trend in dietary exposure. Statistical analyses were mostly descriptive and correlations do not have sufficient sample numbers to be robust. Details of further statistical analyses of room survey data are presented in the Supplementary Information. Where measurements were below limits of detection (LOD) values of LOD\*0.5 have been assumed (median bound).  $\sum \text{BDEs}_{3-7}$  was calculated as the sum of all BDE congeners measured except for BDE-209.

216

## **2.8 Human health risk characterisation**

Potential health risks were calculated from the sum of dust and dietary intake of PBDEs using the margin of exposure (MOE) approach as applied by the European Food Safety Authority (EFSA) for dietary exposure health risk assessment. The MOE is the ratio of the dose at which a small but measureable adverse effect has been reported versus the level of exposure of the population under current consideration. The EFSA Panel on Contaminants in the food chain (EFSA, 2011) identified effects on neurodevelopment as the critical endpoint using BMDL<sub>10</sub> for neurobehavioural effects in mice induced during a relevant period for brain development. Chronic human intakes, associated with body burdens at the BMDL<sub>10</sub> for BDEs-47, -99, -153 and -209, were estimated to be 172, 4.2, 9.6 and 1,700,000 ng kg<sup>-1</sup> bw day<sup>-1</sup> respectively. For PBDEs, EFSA consider that an MOE ratio above 2.5 indicates that a health concern is unlikely, with risk decreasing as the MOE increases (EFSA, 2011). It should be noted that although human intakes of concern are presented as daily doses these represent chronic intake and as such would be better represented as weekly or monthly intakes as daily intakes can be exceeded on occasion without concern as long as other days have lower exposures.

## 234 **3 Results and Discussion**

235 Our cohort consisted of 10 male-female cohabiting couples living in northeast England in  
 236 2011/12. All participants completed full sample and data set collection. Participants were  
 237 recruited from as wide a pool of socio-economic class, occupation, diet and location as  
 238 possible, however, the small number of participants and the focus on breastfeeding  
 239 mothers means that results are not representative of all UK residents' exposures. The  
 240 benefit of the small cohort was that detailed information could be collected for each  
 241 individual allowing the investigation to include almost all contributing factors in PBDE  
 242 exposure known at the time. Further details of occupations, diets, parity, breastfeeding and  
 243 other lifestyle and anthropometric factors are presented in [Supplementary Information](#).  
 244 Previously published serum, breastmilk, and duplicate diet concentrations (Bramwell et al.,  
 245 2014; Bramwell et al., 2016b) have been further examined in this investigation, along with  
 246 new matched dust concentrations, diet and dust intake estimations and exposure and food  
 247 frequency questionnaire, seven day food and activity diary and room survey information in  
 248 order to provide as complete a picture of participants' PBDE exposures as possible.

### 249 **3.1 Dust PBDE concentrations**

250 Dust samples were collected from 40 micro-environments frequently used by the study  
 251 participants. Main living areas (n=10), bedrooms (n=12) and home offices (n=2) were  
 252 sampled. Workplaces were sampled if access was granted by employers (n=8). None of the  
 253 domestic samples were from open plan homes. Four of the workplace samples were from  
 254 open plan indoor spaces. Vehicles were sampled if participants regularly spent more than  
 255 five hours each week in them (n=8). We measured PBDEs in dust from all of the  
 256 microenvironments sampled. Individual concentrations for all PBDEs in each dust sample  
 257 are presented in [Supplementary Information Tables SI 1-4](#) and summaries of the dust  
 258 concentrations in different rooms are presented in [Table 1](#). Median dust  $\Sigma$ BDEs<sub>3-7</sub>  
 259 concentrations were highest in vehicles (179 ng g<sup>-1</sup>) followed by living rooms, bedrooms  
 260 then workplaces (137, 102 and 84 ng g<sup>-1</sup> respectively). Median BDE-209 concentrations in  
 261 dust were also highest in vehicles (19,000 ng g<sup>-1</sup>) then bedrooms, living rooms and  
 262 workplaces (3,530, 2,960, and 2,300 ng g<sup>-1</sup> respectively). The highest concentration of

263  $\Sigma$ BDE<sub>3-7</sub> was measured in a bedroom (7,320 ng g<sup>-1</sup> dust), the highest BDE-183 in the rear of  
264 a work van (367 ng g<sup>-1</sup>) and the highest BDE-209 in a car (137,000 ng g<sup>-1</sup>). Summaries of dust  
265 PBDE concentrations in the different microenvironments are compared with previous UK  
266 and international data in [Table 2](#). Measurements in this study were in keeping with  
267 previously published UK data (Harrad et al., 2008a; Harrad et al., 2008b; Pless-Mulloli et al.,  
268 2006; Sjödin et al., 2008) and in agreement with the theory that BDE-209 usage was greater  
269 in the UK (Fromme et al., 2016; Harrad, 2015). Results were directly comparable to studies  
270 by Harrad et al. (2008a; 2008b) as we used the same sampling protocol, sampling  
271 equipment and laboratory techniques.

272 We compared room survey information such as counts and age of soft furnishings and  
273 electronics and room cleaning frequencies with the concentrations of PBDEs in each room.  
274 Details from individual room surveys are provided in [Supplementary Information Table SI5](#).  
275 We did not find that simple counts of soft furnishings or electronics were good predictors of  
276 high or low PBDE loading. The clearest association between room contents and PBDE  
277 concentrations in dust were for BDE-209 if the room contained a carpet or rugs over 20  
278 years of age (see [Supplementary Information Figure 2](#)). Counts of large PUF items over 20  
279 years old or office chairs from the USA (adhering to Californian state fire retardancy  
280 regulations TB117) correlated significantly with concentrations of Penta mix BDEs only, BDE-  
281 47 ( $r=0.37$ ,  $p=0.036$ ), -99 ( $r=0.35$ ,  $p=0.047$ ) and  $\Sigma$ BDE<sub>3-7</sub> ( $r=0.37$ ,  $p=0.039$ ). Higher dusting  
282 frequency demonstrated the greatest correlation with lower dust PBDE concentrations, with  
283 BDEs-47, -99, -153, -154 and -209 all with correlation significant at the 0.01 level and BDE-  
284 100 with correlation significant at the 0.05 level. [Table SI 6](#) in the [Supplementary](#)  
285 [Information](#) contains further correlation data. Discussion of apparent differences between  
286 repeat sampling weeks' dust data is provided as [Supplementary Information](#).

287 We found that concentrations of  $\Sigma$ Penta product BDEs in the bedroom were significantly  
288 correlated with those in all other environments measured; living rooms ( $r=0.43$ ,  $p=0.05$ ),  
289 workplaces ( $r=0.71$ ,  $p=0.05$ ) and vehicles ( $r=0.90$ ,  $p=0.02$ ). Concentrations of  $\Sigma$ Penta product  
290 BDEs in living room dusts correlated strongly with those in workplaces ( $r=0.90$ ,  $p=0.01$ ) but  
291 not vehicles ( $r=0.30$ ,  $p=0.60$ ). A larger data set may have revealed alternative findings,  
292 particularly for workplaces and vehicles. We suggest that dust particles may briefly adhere  
293 to and then be shaken from skin, hair, clothing and footwear causing distribution among key

294environments used by participants. Further correlation data is provided in Supplementary  
295Information Table SI13.

### 2963.2 Intake of PBDEs via dust

297The ranges of average (20 mg dust ingested d<sup>-1</sup>) and high (50 mg dust ingested d<sup>-1</sup>) PBDE  
298intakes via dust for our study participants was 13.8-1,010 and 35-2,520 pg kg<sup>-1</sup> bw day<sup>-1</sup> for  
299 $\Sigma$ BDEs<sub>3-7</sub>, with 281 to 15,900 and 702 to 39,600 pg kg<sup>-1</sup> bw day<sup>-1</sup> for BDE-209 via dust. Our  
300 $\Sigma$  BDEs<sub>3-7</sub> estimates were similar to previous UK and German  $\Sigma$ BDEs<sub>3-7</sub> intake estimates  
301(Fromme et al., 2009; Harrad et al., 2008a) and an order of magnitude lower than those in  
302the USA (Harrad et al., 2008b). In contrast, our BDE-209 intakes from dust were similar to  
303those of the USA (Harrad et al., 2008b) and an order of magnitude higher than Belgian and  
304German estimates (Fromme et al., 2009; Roosens et al., 2009) (see [Supplementary](#)  
305[Information Table 6](#)). The wide range of intakes reflected the diverse PBDE loadings  
306measured in microenvironment dusts. For this cohort, the influence of specific items in  
307specific microenvironments could be reasonably speculated on a case by case basis.  
308However, although we expected our participant with occupational PUF and furnishing fabric  
309exposure to have a raised PBDE body burden, their fastidious cleaning habits appear to have  
310reduced their exposure.

311The greatest proportion of the estimated dust intake for  $\Sigma$ BDEs<sub>3-7</sub>, BDE-183 and BDE-209  
312took place in the bedroom (means 43%, 38% and 33% respectively) due to the greater  
313amount of time spent in bedrooms. Workplaces and living rooms were the second most  
314important microenvironments for  $\Sigma$ BDEs<sub>3-7</sub> exposure (mean 19%, 13%) and BDE-183 (20%,  
31521%). Vehicles were the second most important microenvironment for BDE-209 intake  
316(20%). The relative proportions of PBDE intakes in different microenvironments for  
317individual participants is illustrated in [Figure 1](#). Our finding that the greater proportion of  
318exposure to all congeners occurs in the bedroom is in keeping with our finding of an  
319association between bedroom dust and serum concentrations of the PBDE congeners found  
320in the commercial Penta-BDE products (BDE-47, -99, -100, -153) ( $r=0.42$ ,  $p=0.04$ ), an  
321association that has also been reported elsewhere (Ali et al., 2014; Coakley et al., 2013;  
322Watkins et al., 2012). Relationships between PBDE in dust and body burdens

We compared PBDE concentrations in dust in the different indoor environments with their matched PBDE body burdens. Significant associations were noted between Penta-mix BDEs in bedroom dust and serum ( $r=0.45$ ,  $p=0.04$ ). BDE-153 in bedroom dust was significantly associated with BDEs-47 ( $r=0.45$ ,  $p=0.03$ ), -99 ( $r=0.45$ ,  $p=0.03$ ), -209 ( $r=0.41$ ,  $p=0.05$ ) and  $\Sigma\text{BDEs}_{3-7}$  ( $r=0.45$ ,  $p=0.03$ ) in serum. BDE-153 in serum was associated but not significantly with BDEs-153 (0.39, 0.06) and  $\Sigma\text{BDEs}_{3-7}$  (0.39, 0.06) in bedroom dust. BDE-47 was associated but not significantly in living room dust and breast milk (0.77, 0.07). BDE-209 was significantly correlated in serum and workplace dusts (0.72, 0.02) however this was strongly influenced by one data point. Also correlated but not significantly in workplace dusts were BDEs-47 (0.57, 0.07) and -99 (0.53, 0.09). [Table SI 7 in Supplementary Information](#) provides further dust and body burden correlation data. No significant correlations were found between vehicle dust and serum despite vehicles having the highest PBDE concentrations in their dust, possibly due to participants spending less time in their cars than in other environments measured. The associations between bedroom dust and serum might be expected due to participants spending the greatest proportion of their day in this room, similarly for associations with workplace dust and serum.

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### 3403.3 Dietary intake of PBDEs

We estimated participants' PBDE intake from diet using three different methods, (i) a 24 hour duplicate diet sample collected the day before taking serum and milk samples, (ii) a seven day food diary completed the seven days prior to serum and milk sampling and (iii) a food frequency questionnaire (FFQ) to represent longer term eating habits. Concentrations of PBDEs in the 24 hour duplicate diet samples summarised in [Table 1](#). BDEs<sub>3-7</sub> were measurable in all of the duplicate diet samples and BDE-209 in 79% of them. 24 hour duplicate diet PBDE concentrations were converted to daily dietary intake estimates which ranged from 82 – 1,320 pg kg<sup>-1</sup> bw for  $\Sigma\text{BDEs}_{3-7}$  and <0.8- 1,860 pg kg<sup>-1</sup> bw for BDE-209. BDE-209 made up a median of 73% of the total PBDE exposure from diet. Estimates of individuals' PBDE intake via diet are provided in [Supplementary Information Table SI 11](#). The mean intake estimates of BDEs-47, -99, -100, -153 and -154 for the omnivores in this study were significantly lower than those measured by Harrad et al. (2004) for duplicate diet samples collected in the West Midlands of the UK in 2002 ( $p=0.01$ ). The 2002 lower bound

mean intakes were within the maximum intakes estimated by this study for BDEs -47, -100, -153 and -154 and upper bound intakes for BDEs -47, -100, and -154. These findings indicate a reduction in dietary exposure during the 10 years between the two studies, with the greatest reductions being for BDE-99 then BDE-153.

Meat, fish and dairy portion consumption estimates compared well between the FFQ and seven day food diaries. Meat portions consumed per week ranged from none to 14 or 15 (FFQ and diary respectively), with median 6.3 or 8 portions. Fish and seafood portions consumed per week ranged from none to 3.5 (maximum for both FFQ and diary), with median 1.8 or 2 portions. Dairy portions consumed per week ranged from none to 25 or 18 (FFQ and diary respectively), with median 8.0 or 8.5 portions. A summary of selected information from the FFQ, diary and 24h duplicate diet is presented in [Table 3](#).

#### **3.4 Relationships between PBDE in diet, serum and breastmilk**

We compared PBDE body burdens with concentrations in the duplicate diet finding a significant association for  $\Sigma$ BDEs<sub>3-7</sub> in both ( $r=0.41$ ,  $p=0.05$ ). Serum samples were collected from fasted participants in order for the serum sample to represent the participants' background PBDE body burden without influence from recently consumed food. Breastmilk samples were not necessarily collected in a fasted state. The complex relationship between historic PBDE deposits in adipose tissue, recent diet, serum and breastmilk is beyond the scope of this paper. We found limited correlation between congeners in serum and breastmilk (see [Supplementary Information Table SI 8](#)), possibly the result of transfer of PBDEs from serum to milk varying between different congeners. Mean serum/milk ratios generally increased with molecular size and hydrophobicity, e.g. 1.3, 3.1 and 6.0 for BDEs-47, -99 and -209. This pattern was in keeping with findings of a 2012 review of PBDE in matched serum and breastmilk samples (Mannetje et al., 2012). BDE-153 in the body appears to follow a different pattern with a serum/milk ratio of 0.4, i.e. more in milk than serum.



We found that the number of meat portions consumed in the week prior to sampling had significant positive correlations with BDEs-99 ( $r=0.46$ ,  $p=0.01$ ) -153 ( $r=0.44$ ,  $p=0.03$ ) and  $\Sigma\text{BDE}_{3-7}$  ( $r=0.43$ ,  $p=0.04$ ) in serum. Further correlation data between dietary information is provided in [Supplementary Information Table SI9](#). The UK FSA 2006 TDS found meat products (followed by fish) to contribute most to the PBDE intake of the general UK population (EFSA, 2011; FSA, 2006). For participants in this study, meat portions consumed exceeded fish portions. Our earlier review of associations between PBDE body burden, dust and diet (Bramwell et al., 2016a) also found eating meat to be the most frequently reported association (eating dairy and fish were next). Similarly, a nationwide study in the USA found vegetarians to have 23% lower, and heavy red meat consumers to have 18% higher total PBDEs in serum than omnivores (Fraser et al., 2009).

393

### 394**3.5 Anthropometric and questionnaire covariates of PBDE body burden**

As well as participants' height, weight and body fat mass measurements, information on travel habits, hand to mouth behaviours, parity, numbers of household members, hobbies and occupations was also collected to look for indicators of higher serum and breast milk PBDE concentrations. These associations are presented in [Supplementary Information Table SI10](#). We found serum BDE-153 concentrations to be significantly associated with sex ( $r=-0.60$ ,  $p=0.01$ ), percentage of body fat mass ( $r=-0.49$ ,  $p=0.02$ ), parity in women ( $r=-0.57$ ,  $p=0.05$ ) and working with electronics ( $r=0.59$ ,  $p=0.01$ ). Males generally had higher BDE-153 in serum than females, in keeping with the findings of a recent Swedish study of 170 adults (Bjermo et al., 2017) and a nationwide study in the USA that found males generally had higher  $\text{BDE}_{3-7}$  body burdens (Fraser et al., 2009). We hypothesise there may be two factors influencing the higher serum concentrations of males in this study, (i) men generally had lower BMI values; seven of the females had recently been pregnant which would increase their BMI and (ii) 9 of the 10 female participants in the study had undergone some depuration effect during pregnancy and breast feeding which their male partners had not. In a study of the breastmilk of 83 women at three and 12 months postpartum, BDE-153 showed a significant increase over time (Daniels et al., 2010) suggesting that BDE-153 present in adipose fat compartments from historic exposures may have been mobilised during the nursing period. Storage of BDE-153 in fat compartments in the body has been

suggested as the reason for dilution in the serum of people with higher BMI (Cequier et al., 2015; Fraser et al., 2009). Why these findings for BDE-153 are not consistent with findings for other congeners is not clear but it may be linked to its longer human half-life (Geyer et al., 2004).

### **3.6 Was diet or dust the major source of PBDE exposure for this cohort?**

Diet was the major source of  $\Sigma\text{BDE}_{3-7}$  for this cohort making up a median of 85% of the total intake when using duplicate diet data with the average dust ingestion estimate of 20 mg d<sup>-1</sup>. This was a somewhat lower proportion than comparable previous studies estimates of 95% (UK), 96% (Belgium) and 97%, (Germany) (Abdallah and Harrad, 2014; Fromme et al., 2009; Roosens et al., 2009) due to our higher median  $\Sigma\text{BDE}_{3-7}$  dust concentration and the notably higher concentration of  $\Sigma\text{BDE}_{3-7}$  in the German duplicate diets (see Table SI 6). We did not include estimates of intake of PBDEs from indoor air in our totals. Previous studies have found PBDE intake from air to constitute <1% of total PBDE intake (Fromme et al., 2009) and a maximum of 2% (Abdallah and Harrad, 2014).

Considering only a cohort's average intake hides the substantial variation between individuals and their exposure sources - something this study has been able to demonstrate clearly (see Figure 2 and Supplementary Information Table SI 6). An individual's total PBDE intake is a combination of dust concentrations in different environments, time spent in them and dietary habits. For example, the proportion of  $\Sigma\text{BDE}_{3-7}$  BDE intake provided by dust for an average dust intake rate had a median 4% but ranged between 0.7% (8M) and 32% (5F). Both these participants lived rurally, the former on a smallholding, the other on a farm. 8M spent the most time outdoors (almost 9 hours each day), had a low Penta-BDE loading in their bedroom dust and, despite a generally home-grown and organic diet, a duplicate diet intake in the 3<sup>rd</sup> quartile. 5F's relatively high dust intake (32% using average dust intake and 54% using high dust intake rates) was due to having the room (bedroom) with the highest  $\Sigma\text{BDE}_{3-7}$  concentrations measured in the study. Although 5F consumed a vegetarian diet their dietary  $\Sigma\text{BDE}_{3-7}$  intake was in the top quartile.

Dust was the greatest source of BDE-209 for our entire cohort, with median intakes making up 75% and 88% of the total BDE-209 intake for average and high dust intake rates

443respectively, lower than previous UK estimates of 94% and 99% (Abdallah and Harrad, 2014;  
444Harrad, 2010) possibly due to declining use of Deca-BDE product and differences between  
445cohorts in the different studies. Individual participants' proportion of total BDE-209 intake  
446provided by dust for average dust intake rate ranged from 14% (8M) to 100% (1Fii and  
4471Mii). Participant 10M had a significantly greater BDE-209 concentration than their partner  
448possibly a reflection of the relatively high amount of time spent in their vehicle (23% of their  
449time) and BDE-209 concentration in their car (30,338 ng/g).

450We found the range of individuals' intakes of  $\Sigma\text{BDEs}_{3-7}$  from dust to be five times greater  
451than their intakes from diet. The highest total intake (using average dust intake scenario)  
452was 16 times greater than the lowest reported intake. Our data agrees with previous  
453hypotheses that the wide range in PBDE concentrations in room dusts (compared with the  
454range seen in diets) may be the reason some individuals have significantly higher internal  
455dose (Harrad et al., 2008b; Petreas et al., 2003; Thomas et al., 2006; Wu et al., 2007). Dust  
456generation, dust ingestion rates, and cleaning frequencies (both microenvironments and  
457hand washing) may also be influential.

458Our study corroborates previous studies findings that average PBDE intakes in the UK are broadly  
459similar to those in mainland Europe, where meat is the major source of Penta-BDEs for the average  
460person but dust is the major source of BDE-209 (Bramwell et al., 2016a; Harrad et al., 2008b). For  
461infants, the average contribution to total intakes from diet were >90% for  $\Sigma\text{BDEs}_{3-7}$  and 69% for BDE-  
462209. At the high dust ingestion rate this decreased to 35-50% for  $\Sigma\text{BDEs}_{3-7}$  and 88% for BDE-209.  
463These figures indicate similar proportional intake for infants from diet to our adults, although with  
464considerably higher amounts ingested per kg body weight (see [Table 3](#)).

465

### 466**3.7 Study Limitations**

467This study involved a relatively small cohort of 20 individuals (10 UK couples). The study philosophy  
468concentrated more on the details and habits of the volunteers in order to understand their  
469individual exposures. The volume of usage of PBDE mixtures such as PentaBDE, the timelines of  
470product introduction and restriction, either voluntary or regulation enforced, and the type of usage,  
471are all variables in general population exposure. For example, a far greater volume of the PentaBDE  
472mixture was used in the USA and Canada compared to Europe and this is reflected in the relatively  
473higher concentrations of related congeners measured in serum, and in house dust levels from North

474America. Also, where we found diet to be the most important exposure pathway for Penta mix BDEs,  
475studies such as (Lorber, 2008) have shown that dust is a major pathway for PentaBDE in North  
476American populations. When personal details and habits are considered, the exposure assessment  
477is even more unique. Thus, the finding of this study are not intended to be representative of the UK  
478as a whole, or even less, other regions of the world.

479

### 480**3.8 Risk characterisation**

481The most relevant congener from a health risk perspective is BDE-99 but there is no  
482agreement on a safe intake. The US-EPA suggests a reference dose 100 ng/kg bw/day (US-  
483EPA, 2006) whereas the more recent EFSA suggested health reference value is 4.2 ng/kg  
484bw/day with an MOE of 2.5 (EFSA, 2011). We investigated potential health risk from our  
485estimated PBDE intakes by comparing them with both these reference values (see [Table 4](#)  
486and [Table SI12](#)). The combined uncertainties from household types, sampling and  
487measurement is likely be quite high and should be borne in mind. No health concerns are  
488expected from the PBDE intakes estimated in this study for adults as all had MOEs over 2.5  
489(EFSA, 2011). The lowest adult MOEs were 2.8 and 3.7 for BDE-99 using a high dust intake  
490rate for household 5 with the high BDE<sub>3-7</sub> measurements in their bedroom. Accordingly,  
491estimated infant daily exposures to BDE-99 for the same home have MOEs below those  
492recommended by EFSA for chronic exposure. Using average diet intake data from the 2012  
493UK TDS with dust exposure data from this study with average dust intake rates we found the  
494lowest MOE estimation to be 2.3 which is similar to the EFSA recommended MOE of 2.5  
495deemed to indicate a potential health risk. Using high dust intake rates with dust data for  
496this study and 97.5<sup>th</sup> percentile (P97.5) dietary intake estimates from the 2012 UK TDS this  
497MOE dropped to 0.7 and two additional homes indicated high infant intake MOEs between  
4982.5 and 3. All other adult and infant MOEs using EFSA reference values and all MOEs using  
499US EPA values were comfortably above the recommended MOE. Follow-up measurement of  
500the PBDE body burdens for infants of parents participating in this study could help describe  
501associations with raised intake estimations.

502

## 503 4 Conclusions

504 This detailed study is the first anywhere to document concentrations of PBDEs, including  
505 BDE-209, in samples of indoor dust and diet with matched human serum and breast milk  
506 concentrations. Our findings confirmed that both diet and dust make a contribution to PBDE  
507 body burdens and provide new evidence of a wide range in their relative contributions  
508 between individuals. Diet appeared to be the primary source of intake of BDE<sub>3-7</sub> congeners  
509 for the majority of this cohort, and meat consumption demonstrated the strongest  
510 significant positive association between diet type and serum BDE<sub>3-7</sub> concentrations. Dust  
511 was the cohort's primary source of BDE-209. Rooms containing a carpet or rugs over 20  
512 years old had higher BDE-209 concentrations in their dust. Rooms that were dusted more  
513 frequently had less BDE-209, as well as less Penta mix PBDE congeners. Rooms containing  
514 sofas or armchairs over 20 years old had higher concentrations of commercial Penta mix  
515 PBDE congeners. BDE-209 concentrations in room dusts did not widely correlate with BDE-  
516 209 body burdens, possibly due to the congener's relatively large molecular size and low  
517 bioaccessibility. Correlations between BDE<sub>3-7</sub> congeners in serum and indoor dust were  
518 strongest in bedrooms in keeping with the greater proportion of time spent there. Being  
519 male and having a lower body fat mass were indicators of higher serum BDE-153 for this  
520 cohort. BDE-99 was the congener demonstrating the lowest MOE (and therefore the  
521 greatest health risk) and although we found a reduction in dietary exposure to this and  
522 other Penta-mix PBDEs since 2002, reducing dietary exposure would still have the greatest  
523 effect in reducing body burdens.

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530

531 **Conflicts of interest:** None

532

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